Stress-associated cardiovascular reaction masks heart rate dependence on physical load in mice

Titre	Stress-associated cardiovascular reaction masks heart rate dependence on physical load in mice
Type de publication	Article de revue
Auteur	Andreev-Andrievskiy, A. A [1], Popova, A. S [2], Borovik, A. S [3], Dolgov, O. N [4], Tsvirkun, D. V [5], Custaud, Marc-Antoine [6], Vinogradova, O. L [7]
Editeur	Elsevier
Type	Article scientifique dans une revue à comité de lecture
Année	2014
Langue	Anglais
Date	2014
Pagination	1 - 9
Volume	132
Titre de la revue	Physiology & Behavior
ISSN	1873-507X
Résumé en anglais	When tested on the treadmill mice do not display a graded increase of heart rate (HR), but rather a sharp shift of cardiovascular indices to high levels at the onset of locomotion. We hypothesized that under test conditions cardiovascular reaction to physical load in mice is masked with stress-associated HR increase. To test this hypothesis we monitored mean arterial pressure (MAP) and heart rate in C57BL/6 mice after exposure to stressful stimuli, during spontaneous locomotion in the open-field test, treadmill running or running in a wheel installed in the home cage. Mice were treated with beta1-adrenoblocker atenolol (2mg/kg ip, A), cholinolytic ipratropium bromide (2mg/kg ip, I), combination of blockers (A+I), anxiolytic diazepam (5mg/kg ip, D) or saline (control trials, SAL). MAP and HR in mice increased sharply after handling, despite 3weeks of habituation to the procedure. Under stressful conditions of open field test cardiovascular parameters in mice were elevated and did not depend on movement speed. HR values did not differ in I and SAL groups and were reduced with A or A+I. HR was lower at rest in D pretreated mice. In the treadmill test HR increase over speeds of 6, 12 and 18m/min was roughly 1/7-1/10 of HR increase observed after placing the mice on the treadmill. HR could not be increased with cholinolytic (I), but was reduced after sympatholytic (A) or A+I treatment. Anxiolytic (D) reduced heart rate at lower speeds of movement and its overall effect was to unmask the dependency of HR on running speed. During voluntary running in non-stressful conditions of the home cage HR in mice linearly increased with increasing running speeds. We conclude that in test situations cardiovascular reactions in mice are governed predominantly by stress-associated sympathetic activation, rendering efforts to evaluate HR and MAP reactions to workload unreliable.
URL de la notice	http://okina.univ-angers.fr/publications/ua8422 [8]

DOI	10.1016/j.physbeh.2014.03.033 [9]
Lien vers le document	http://dx.doi.org/10.1016/j.physbeh.2014.03.033 [9]
Titre abrégé	Physiol Behav

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